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Search Results -

Terms	Documents
L10 and diagnosis	27

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Refine Search: L10 and diagnosis Clear

Search History

Today's Date: 10/15/2001

DB Name	Query	Hit Count	Set Name
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L10 and diagnosis	27	L11
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L8 and diabetes	61	L10
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L8 and diabet?	0	L9
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L3 and insulin	133	L8
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L3 and insulin?	0	L7
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L3 and (insulin? or diabet?)	0	L6
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	keratinocyte? and (FABP?)	9	L5
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	keratinocyte? and (FABP)	11	L4
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	keratinocyte? and (fatty adj acid?)	491	L3
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	Mal1?	3	L2
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	Mal1	12	L1

Day : Monday
Date: 10/15/2001
Time: 14:39:37

PALM INTRANET

Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	First Name	
hotamisligil	gokhan	Search

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Back to || PALM || ASSIGNMENT || OASIS || Home Page

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PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

search result for 09/788,074 NON-final 1st Rej.

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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Dec 17	The CA Lexicon available in the CAPLUS and CA files
NEWS 3	Feb 06	Engineering Information Encompass files have new names
NEWS 4	Feb 16	TOXLINE no longer being updated
NEWS 5	Apr 23	Search Derwent WPINDEX by chemical structure
NEWS 6	Apr 23	PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
NEWS 7	May 07	DGENE Reload
NEWS 8	Jun 20	Published patent applications (A1) are now in USPATFULL
NEWS 9	JUL 13	New SDI alert frequency now available in Derwent's DWPI and DPCI
NEWS 10	Aug 23	In-process records and more frequent updates now in MEDLINE
NEWS 11	Aug 23	PAGE IMAGES FOR 1947-1966 RECORDS IN CAPLUS AND CA
NEWS 12	Aug 23	Adis Newsletters (ADISNEWS) now available on STN
NEWS 13	Sep 17	IMSworld Pharmaceutical Company Directory name change to PHARMASEARCH
NEWS 14	Oct 09	Korean abstracts now included in Derwent World Patents Index
NEWS 15	Oct 09	Number of Derwent World Patents Index updates increased
NEWS 16	Oct 15	Calculated properties now in the REGISTRY/ZREGISTRY File

NEWS EXPRESS	August 15 CURRENT WINDOWS VERSION IS V6.0c, CURRENT MACINTOSH VERSION IS V6.0 (ENG) AND V6.0J (JP), AND CURRENT DISCOVER FILE IS DATED 07 AUGUST 2001
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

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FILE 'HOME' ENTERED AT 14:24:28 ON 15 OCT 2001

=> file medline embase caplus scisearch biosis reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.15	0.15

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FILE 'EMBASE' ENTERED AT 14:24:40 ON 15 OCT 2001
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PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2001 American Chemical Society (ACS)

=> s Mal1
L1          259 MAL1

=> s (keratinocyte adj fatty adj acid)
L2            0 (KERATINOCYTE ADJ FATTY ADJ ACID)

=> s keratinocyte
L3        76939 KERATINOCYTE

=> s l3 and (fatty adj acid?)
L4            0 L3 AND (FATTY ADJ ACID?)

=> s l3 and FABP?
L5          129 L3 AND FABP?

=> s L3 and (binding adj protein?)
   5 FILES SEARCHED...
L6            0 L3 AND (BINDING ADJ PROTEIN?)

=> s l1 and (insulin? or diabet?)
L7            2 L1 AND (INSULIN? OR DIABET?)

=> s L5 and (insulin? or diabet?)
L8            9 L5 AND (INSULIN? OR DIABET?)

=> s l7 or l8
L9           10 L7 OR L8

=> dup rem l9
DUPLICATE IS NOT AVAILABLE IN 'REGISTRY'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L9
L10           6 DUP REM L9 (4 DUPLICATES REMOVED)

=> d l10 ibib abs 1-
YOU HAVE REQUESTED DATA FROM 6 ANSWERS - CONTINUE? Y/(N):y

L10  ANSWER 1 OF 6  CAPLUS  COPYRIGHT 2001 ACS
```

ACCESSION NUMBER: 2001:617839 CAPLUS
DOCUMENT NUMBER: 135:200396
TITLE: Treatment lipid metabolic disorders by inhibiting **Mal1**, the keratinocyte lipid binding protein activity
INVENTOR(S): Hotamisligil, Gokhan S.
PATENT ASSIGNEE(S): President and Fellows of Harvard College, USA
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001060384	A1	20010823	WO 2001-US5019	20010216

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-183106 P 20000217

AB The invention is based on the discovery that decreasing **Mal1** (also called keratinocyte fatty acid binding protein) expression prevents or inhibits the development of obesity, **insulin** resistance, **diabetes**, dyslipidemia, and atherosclerosis. Accordingly, the invention features a method of preventing or inhibiting such conditions by administering to a mammal, who has been identified as suffering from or at risk of developing one or more of the above-listed pathologies, a compd. that reduces expression or activity of **Mal1**. Preferably, the compd. inhibits transcription of endogenous **Mal1** by binding to a cis-acting regulatory sequence of the **Mal1** gene and decreases **Mal1** transcription. Alternatively, the compd. inhibits translation of **Mal1** mRNA into a **Mal1** gene product by antisense therapy carried out by administering a single stranded nucleic acid complementary at least a portion of **Mal1** mRNA. The invention also includes a method of preventing or inhibiting the development of obesity, **insulin** resistance, **diabetes**, dyslipidemia, and atherosclerosis by administering to a mammal a compd. that reduces activity of **Mal1**. The invention also includes a method of diagnosing individuals who are at risk of developing lipid metabolic disorders.

REFERENCE COUNT: 6
REFERENCE(S):
(1) Hertzel, A; GENE 1998, V221(2), P235 CAPLUS
(2) Hotamisligil, G; WO 0047734 A 2000 CAPLUS
(3) Hotamisligil, G; SCIENCE 1996, V274(5291), P1377 CAPLUS
(4) Kane, C; BIOCHEMISTRY 1996, V35(9), P2894 CAPLUS
(6) Shaughnessy, S; DIABETES 2000, V49(6), P904 CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 6 MEDLINE DUPLICATE 1

ACCESSION NUMBER: 2001103644 MEDLINE
DOCUMENT NUMBER: 20515662 PubMed ID: 11060343
TITLE: Lipid-binding proteins modulate ligand-dependent trans-activation by peroxisome proliferator-activated receptors and localize to the nucleus as well as the cytoplasm.
AUTHOR: Helledie T; Antonius M; Sorensen R V; Hertzel A V; Bernlohr D A; Kolvraa S; Kristiansen K; Mandrup S
CORPORATE SOURCE: Department of Molecular Biology, University of Southern Denmark, Odense, DK-5230 Odense M, Denmark.
SOURCE: JOURNAL OF LIPID RESEARCH, (2000 Nov) 41 (11) 1740-51. Journal code: IX3. ISSN: 0022-2275.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010208

AB Peroxisome proliferator-activated receptors (PPARs) are activated by a variety of fatty acids, eicosanoids, and hypolipidemic and **insulin**-sensitizing drugs. Many of these compounds bind avidly to members of a family of small lipid-binding proteins, the fatty acid-binding proteins (**FABPs**). Fatty acids are activated to CoA esters, which bind with high affinity to the acyl-CoA-binding protein (ACBP). Thus, the availability of known and potential PPAR ligands may be regulated by lipid-binding proteins. In this report we show by transient transfection of CV-1 cells that coexpression of ACBP and adipocyte lipid-binding protein (ALBP) exerts a ligand- and PPAR subtype-specific attenuation of PPAR-mediated trans-activation, suggesting that lipid-binding proteins, when expressed at high levels, may function as negative regulators of PPAR activation by certain ligands. Expression of ACBP, ALBP, and **keratinocyte** lipid-binding protein (KLBP) is induced during adipocyte differentiation, a process during which PPARgamma plays a prominent role. We present evidence that endogenous ACBP, ALBP, and KLBP not only localize to the cytoplasm but also exhibit a prominent nuclear localization in 3T3-L1 adipocytes. In addition, forced expression of ACBP, ALBP, and KLBP in CV-1 cells resulted in a substantial accumulation of all three proteins in the nucleus. These results suggest that lipid-binding proteins, contrary to the general assumption, may exert their action in the nucleus as well as in the cytoplasm.

L10 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:647848 CAPLUS
DOCUMENT NUMBER: 134:351081
TITLE: Adipocyte metabolism in adipocyte fatty acid binding protein knockout (aP2-/-) mice after short-term high-fat feeding: functional compensation by the **keratinocyte** [sic] fatty acid binding protein. [Erratum to document cited in CA133:132923]
AUTHOR(S): Shaughnessy, Sara; Smith, Elizabeth R.; Kodukula, Sarala; Storch, Judith; Fried, Susan K.
CORPORATE SOURCE: Department of Nutritional Sciences, Rutgers University, New Brunswick, NJ, 08901-8525, USA
SOURCE: Diabetes (2000), 49(9), 1617

CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The word "**keratinocyte**" was incorrectly spelled as "keritinocyte".
REFERENCE COUNT: 3
REFERENCE(S): (1) Mammes, O; Diabetes 1998, V47, P487 CAPLUS
(2) Shaughnessy, S; Diabetes 2000, V49, P904 CAPLUS
(3) Virtanen, S; Diabetes 2000, V49, P912 CAPLUS

L10 ANSWER 4 OF 6 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 2000:564381 SCISEARCH
THE GENUINE ARTICLE: 335GZ
TITLE: Adenovirus-mediated gene transfer in primary murine adipocytes
AUTHOR: Hertzel A V; Sanders M A; Bernlohr D A (Reprint)
CORPORATE SOURCE: UNIV MINNESOTA, DEPT BIOCHEM MOL BIOL & BIOPHYS, ST PAUL, MN 55108 (Reprint); UNIV MINNESOTA, DEPT BIOCHEM MOL BIOL & BIOPHYS, ST PAUL, MN 55108; UNIV MINNESOTA, IMAGING CTR,
ST PAUL, MN 55108
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF LIPID RESEARCH, (JUL 2000) Vol. 41, No. 7, pp. 1082-1086.
Publisher: LIPID RESEARCH INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998.
ISSN: 0022-2275.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 11
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The transfer of genes into primary murine adipocytes using an adenovirus system has been developed. A recombinant adenovirus was constructed (expressing green fluorescent protein [GFP] under the control of the strong cytomegalovirus [CMV] promoter and a luciferase reporter gene under the control of the weak adipocyte promoter **keratinocyte** lipid-binding protein [KLBP/**FABP5**]) and incubated with primary adipocytes from C57BL/6J mice. Analysis of infected cells by confocal microscopy detected GFP expression in both the cytoplasm and nucleus of adipocytes with a 64% efficiency of infection. To demonstrate the applicability of this method in the study of gene regulation, adenovirus-infected adipocytes exhibited significant levels of luciferase activity even from a weak promoter. TPA treatment of infected adipocytes increased luciferase activity, consistent with previous studies indicating that the KLBP/ **FABP5** gene is up-regulated by phorbol esters. These results provide an efficient, convenient, and sensitive method to transiently infect primary murine adipocytes, facilitating protein expression or permitting analysis of reporter gene activity from both viral and endogenous promoters.

L10 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:398680 CAPLUS
DOCUMENT NUMBER: 133:132923
TITLE: Adipocyte metabolism in adipocyte fatty acid binding protein knockout (aP2-/-) mice after short-term high-fat feeding: functional compensation by the **keratinocyte** fatty acid binding protein

AUTHOR(S): Shaughnessy, Sara; Smith, Elizabeth R.; Kodukula, Sarala; Storch, Judith; Fried, Susan K.
CORPORATE SOURCE: Department of Nutritional Sciences, Rutgers University, New Brunswick, NJ, 08901-8525, USA
SOURCE: Diabetes (2000), 49(6), 904-911
CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Mice null for adipocyte fatty acid binding protein (AFABP) compensate by increasing expression of **keratinocyte** fatty acid binding protein (KFABP). In the present study, AFABP knockout (KO) and wild-type (WT) mice became equally obese on a high-fat diet, as judged by fat pad wts., adipocyte size, and body compn. anal. High-fat feeding led to moderate **insulin** resistance in both WT and AFABP knockout mice, as indicated by an .apprx.2-fold increase in plasma **insulin**. However, in the high fat-fed mice, plasma glucose levels were .apprx.15% lower in the AFABP-KO mice. Adipocytes isolated from AFABP-KO and WT mice fed high- or low-fat diets exhibited similar rates of basal and norepinephrine-stimulated lipolysis and **insulin**-stimulated rates of glucose conversion to fatty acids and glyceride-glycerol. However, basal glucose conversion to fatty acids was higher in adipocytes of AFABP-KO mice. Adipocyte tumor necrosis factor-.alpha. release was similarly increased by high-fat diet-induced obesity in both WT and AFABP-KO mice. As assessed by Western blot anal., the level of KFABP protein in AFABP-KOs was .apprx.40% of the level of AFABP in WT controls. The binding affinities of KFABP for long-chain fatty acids were 2- to 4-fold higher than those of AFABP, but the relative affinities for different fatty acids were similar. As for AFABP, the rate of fatty acid transfer from KFABP to model phospholipid vesicles was increased with acceptor membrane concn. and by inclusion of acidic phospholipids, indicating a similar mechanism of transfer. We conclude KFABP can functionally compensate for the absence of AFABP, resulting in no major alterations in adipocyte metab. or fat accumulation in response to short-term feeding of high-fat diets that result in moderate hyperinsulinemia.

REFERENCE COUNT: 24
REFERENCE(S):
(1) Binas, B; FASEB J 1999, V13, P805 CAPLUS
(2) Coe, N; J Lipid Res 1999, V40, P967 CAPLUS
(3) DiGirolamo, M; J Lipid Res 1974, V15, P332 CAPLUS
(4) Dole, V; J Biol Chem 1960, V235, P2595 CAPLUS
(5) Edens, N; Am J Physiol 1993, V265, PE374 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 6 OF 6 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 94371631 EMBASE
DOCUMENT NUMBER: 1994371631
TITLE: Retinoic acid induces expression of PA-**FABP** (psoriasis-associated fatty acid-binding protein) gene in human skin in vivo but not in cultured skin cells.
AUTHOR: Larsen F.G.; Voorhees J.J.; Astrom A.
CORPORATE SOURCE: Department of Dermatology, Bispebjerg Hospital,2400 Copenhagen NV, Denmark
SOURCE: Experimental Dermatology, (1994) 3/5 (212-218).
ISSN: 0906-6705 CODEN: EXDEEY
COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology
037 Drug Literature Index

LANGUAGE: English
SUMMARY LANGUAGE: English
AB PA-**FABP** (psoriasis-associated fatty acid-binding protein) is a new member of a group of low-molecular-weight proteins that are highly up-regulated in psoriatic skin and that share similarity to fatty acid-binding proteins. In this study we demonstrate that PA-**FABP** transcripts are expressed in human skin in vivo and that topical application of 0.05% retinoic acid (RA) cream results in a rapid induction of PA-**FABP** transcripts following treatment for 16 hours and persists at increasing levels after 48 and 96 h of RA treatment. The PA-**FABP** mRNA response to RA was reduced by approximately 50% when patients concurrently were treated with RA and 0.025% clobetasol propionate (CLO) for 48 and 96 h, whereas treatment with CLO alone resulted in PA-**FABP** transcript levels not significantly different from vehicle-treated skin. When comparing the effects of a well-known irritant, sodium lauryl sulfate (SLS), to those of RA and its vehicle, 0.05% RA cream but not 2% SLS in RA vehicle caused PA-**FABP** mRNA induction after 16 h. SLS treatment of human skin for 96 h caused a slight increase in PA-**FABP** transcripts, but markedly less than that observed in response to RA treatment. Incubation of cultured human **keratinocytes** or skin fibroblasts with RA for up to 48 h did not significantly induce PA-**FABP** transcripts. Expression of PA-**FABP** message in **keratinocytes** was observed to be induced by calcium and fetal calf serum (FCS), while tetra-decanoyl phorbol acetate (TPA) caused little or no induction. Taken together, the marked inducibility of the PA-**FABP** gene is compatible with the possibility that this gene might be important in RA-mediated regulation of human skin growth and differentiation.

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 NEWS  5  Apr 23   Search Derwent WPINDEX by chemical structure
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 NEWS  7  May 07   DGENE Reload
 NEWS  8  Jun 20   Published patent applications (A1) are now in USPATFULL
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 NEWS 11  Aug 23   PAGE IMAGES FOR 1947-1966 RECORDS IN CAPLUS AND CA
 NEWS 12  Aug 23   Adis Newsletters (ADISNEWS) now available on STN
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                   to PHARMASEARCH
 NEWS 14  Oct 09   Korean abstracts now included in Derwent World Patents
                   Index
 NEWS 15  Oct 09   Number of Derwent World Patents Index updates increased
 NEWS 16  Oct 15   Calculated properties now in the REGISTRY/ZREGISTRY File

 NEWS EXPRESS  August 15 CURRENT WINDOWS VERSION IS V6.0c,
               CURRENT MACINTOSH VERSION IS V6.0 (ENG) AND V6.0J (JP),
               AND CURRENT DISCOVER FILE IS DATED 07 AUGUST 2001
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=> file medline embase caplus scisearch biosis reg
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                 0.15       0.15
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FILE 'MEDLINE' ENTERED AT 15:10:32 ON 15 OCT 2001

FILE 'EMBASE' ENTERED AT 15:10:32 ON 15 OCT 2001

FILE 'CAPLUS' ENTERED AT 15:10:32 ON 15 OCT 2001
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FILE 'REGISTRY' ENTERED AT 15:10:32 ON 15 OCT 2001
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```
=> s diabetes or (insulin adj resistance)
L1      592865 DIABETES OR (INSULIN ADJ RESISTANCE)

=> s l1 and diagnosis
L2       71359 L1 AND DIAGNOSIS

=> s l2 and transcript?
L3         336 L2 AND TRANSCRIPT?

=> s l2 and Mal1
L4           1 L2 AND MAL1

=> s l2 and (keratinocyte adj fatty adj acid?)
   5 FILES SEARCHED...
L5           0 L2 AND (KERATINOCYTE ADJ FATTY ADJ ACID?)

=> d l4 ibib abs 1-
YOU HAVE REQUESTED DATA FROM 1 ANSWERS - CONTINUE? Y/(N):y

L4   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2001 ACS
ACCESSION NUMBER:        2001:617839  CAPLUS
DOCUMENT NUMBER:         135:200396
TITLE:                   Treatment lipid metabolic disorders by inhibiting
                         Mal1, the keratinocyte lipid binding protein
                         activity
INVENTOR(S):             Hotamisligil, Gokhan S.
PATENT ASSIGNEE(S):      President and Fellows of Harvard College, USA
SOURCE:                  PCT Int. Appl., 27 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ---------------   ----  --------      ---------------  --------
     WO 2001060384      A1   20010823      WO 2001-US5019   20010216
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
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CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: US 2000-183106 P 20000217
AB The invention is based on the discovery that decreasing **Mal1** (also called keratinocyte fatty acid binding protein) expression prevents or inhibits the development of obesity, insulin resistance, **diabetes**, dyslipidemia, and atherosclerosis. Accordingly, the invention features a method of preventing or inhibiting such conditions by administering to a mammal, who has been identified as suffering from or at risk of developing one or more of the above-listed pathologies, a compd. that reduces expression or activity of **Mal1**. Preferably, the compd. inhibits transcription of endogenous **Mal1** by binding to a cis-acting regulatory sequence of the **Mal1** gene and decreases **Mal1** transcription. Alternatively, the compd. inhibits translation of **Mal1** mRNA into a **Mal1** gene product by antisense therapy carried out by administering a single stranded nucleic acid complementary at least a portion of **Mal1** mRNA. The invention also includes a method of preventing or inhibiting the development of obesity, insulin resistance, **diabetes**, dyslipidemia, and atherosclerosis by administering to a mammal a compd. that reduces activity of **Mal1**. The invention also includes a method of diagnosing individuals who are at risk of developing lipid metabolic disorders.
REFERENCE COUNT: 6
REFERENCE(S): (1) Hertzel, A; GENE 1998, V221(2), P235 CAPLUS
(2) Hotamisligil, G; WO 0047734 A 2000 CAPLUS
(3) Hotamisligil, G; SCIENCE 1996, V274(5291), P1377 CAPLUS
(4) Kane, C; BIOCHEMISTRY 1996, V35(9), P2894 CAPLUS
(6) Shaughnessy, S; DIABETES 2000, V49(6), P904 CAPLUS
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---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	70.18	70.33

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION

CA SUBSCRIBER PRICE -0.59 -0.59

* *

Dear valued customer,

Your feedback is important to us. Would you kindly take a moment to complete our survey? This survey will only take about 5-10 minutes to complete. Your responses will be kept confidential and will help us improve STN Express with Discover! for your future use. Please click on the following link to access the survey.

http://www.cas.org/ONLINE/STN/ExpressSurveyForm.html?LOGINID=SSSPTA1635LAN

* *

STN INTERNATIONAL LOGOFF AT 15:13:21 ON 15 OCT 2001